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Characterization of Structural and Mechanical Properties of IPSC-derived Cardiomyocytes for Future Application to Familial Cardiomyopathies

Paige Cloonan
Washington University in St. Louis

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Mentor: Michael Greenberg

Familial cardiomyopathies are a leading cause of sudden death in young people, and as of now, there is no known cure. While mouse models and patient heart tissue have been extraordinarily useful in the study of the disease pathogenesis, the physiological differences between mouse and human cardiomyocytes can introduce many inconsistencies in disease presentation. Additionally, tissue from patients with the disease is difficult to obtain and it has undergone many compensations that make it difficult to understand the early stages of the disease pathogenesis. To develop a new model of the disease, we are using human stem cell derived cardiomyocytes bearing mutations that cause familial hypertrophic cardiomyopathies. We have introduced these mutations using CRISPR. Wild type stem cells were differentiated into cardiomyocytes. Immunostaining helped assess sarcomeric organization and traction force microscopy provided a tool to measure the force produced by single cardiomyocytes. We are now beginning to apply the same tools to study a hypertrophic cardiomyopathy disease causing mutation in troponin T, R92Q.